

Modern Concepts of Cardiovascular Disease

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SURGICAL CONSIDERATIONS OF ACQUIRED DISEASES OF THE AORTA AND MAJOR PERIPHERAL ARTERIES*†

II. Dissecting Aneurysms of the Aorta**

Dissecting aneurysm of the aorta is a distinct clinical and pathological entity, characterized by hemorrhagic intramural separation of the aortic wall in the region of the media, usually communicating with the normal lumen by an intimal tear. Males are affected about twice as often as females, with the highest age incidence in the fourth to seventh decades. The etiology of the disease remains obscure, although certain factors have been considered to have a causal relationship owing to their frequent association with its occurrence. These include arachnodactyly (Marfan's syndrome), pregnancy, hypertension, coarctation, and idiopathic kyphoscoliosis.

The underlying predominant lesion appears to be degeneration of the elements of the media, whether or not it is to be considered a form of the classic "medionecrosis cystica" of Fordheim or of "faults" of Shennan. It is apparently unrelated to atherosclerosis or syphilis. The pathogenesis of the initial rupture or tear in the intima and media is not well understood.

According to the prevailing hypothesis, the lesion develops as an intramural hematoma from rupture of the thin-walled vasa vasorum within an area of focal medionecrosis, with secondary rupture of the intima. The dissection commonly begins as a transverse tear in the intima and media a few centimeters above the aortic valve, in the descending thoracic aorta near the origin of the left subclavian artery, or near the ligamentum arteriosum. Once this occurs, separation of the intramural layers of the aorta by the forceful stream of blood produces dissection, usually at the junction of the middle and outer thirds of the media. The dissection may then progress circumferentially to involve all or a portion of the aorta and move distally down to the bifurcation and even lower to invade the femoral arteries. As branches are encountered, they may be sheared off or the dissecting process

may extend along them for varying distances, thus diminishing or completely interrupting the blood supply to these areas. The extent and course of this process are variable. In the most acute and severe forms, dissection is rapid and finally terminal perforation occurs through the adventitia into the pericardium, mediastinum, pleural or peritoneal cavities, with death in a few hours or days. The subacute type may begin abruptly and then progress gradually over a period of days or weeks to terminal adventitial rupture and death. In still more chronic forms of the disease, the defective passage may re-enter the true lumen of the aorta to form a "double-barreled" aorta. The false process may then become covered with an endothelium or be obliterated by the formation of thrombi and subsequent fibrous tissue organization.

Clinical Manifestations

Owing to the variation in extent and progression of the dissecting process, the clinical manifestations may be protean, with symptoms and signs occurring first in the chest and extending more or less rapidly to other regions such as the back, head, neck, shoulders, abdomen, groin, and lower extremities. This sequence or "marching" of manifestations from one region to another is a reflection of the progress of the dissection and the consequent injurious or ischemic effects upon the organs supplied by the major arterial branches of the aorta. The most striking

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** Part I of this article, concerned with aortic aneurysms, appeared in the October issue. Part III, "Atherosclerotic Occlusive Vascular Disease," will be published in January.

Table I

Dissecting Aneurysms of Aorta—Operative Mortality

Blood Pressure	Number of Cases	Deaths	
		Number	Per Cent
149/89 or less	11	0	
150/90 or more	34	11	32
Total	45	11	24

manifestation of the disease is the sudden onset of severe, almost indescribable pain in the chest or epigastrium, which may extend along the course of the ribs to the back and then up into the neck and shoulders or down into the abdomen. In some instances, the onset may be marked by loss of consciousness; in others, shock with significant lowering of blood pressure is frequently present. Depending upon the degree of interference with the blood supply of the central nervous system, abdominal viscera, or extremities, a variety of neurological, abdominal, or peripheral manifestations may develop.

The most important factor in the diagnosis of dissecting aneurysm is awareness of its occurrence, particularly in the presence of the characteristic clinical manifestations. In its early stages, the clinical picture is most often confused with myocardial infarction, but in most cases of dissecting aneurysm the electrocardiogram shows either a normal pattern or only evidence of left ventricular hypertrophy. Characteristically, roentgenograms of the chest show widening and radiolucence of the aortic arch and descending thoracic aorta. The most common finding is widening of the supracardiac shadow involving the entire thoracic aorta, or some segment of it. This may be better evaluated by comparison with roentgenograms of the chest taken prior to the dissection. A double aortic shadow, consisting of a somewhat dense central core and an outer more radiolucent portion representing the dissected passage, is a common radiological finding. Angiocardiography is probably the most valuable diagnostic procedure. In this examination, the contrast agent appears heavily concentrated in the true aortic lumen, whereas a false passage appears as a lateral or medial, somewhat superimposed, less dense channel.

The prognosis in dissecting aneurysms of the aorta is extremely grave. In the classic study by Shennan,* for example, death occurred within 24 hours after onset in 58 per cent of the patients, and after one day to one week in 26 per cent. Death may occur rapidly from rupture into the pleural, pericardial, or peritoneal cavities, or more slowly from progressive mediastinal or retroperitoneal hemorrhage, cardiac failure or renal failure. In only about 10 to 15 per cent of patients with the chronic or healed type of dissection is there a significant chance of survival. Even in this group, further dissection may occur, or death may result from cardiac failure.

Treatment

During the past few years, two methods of surgical treatment have been devised for this condition, depending upon the site and extent

of the dissecting process. The first is indicated in cases in which the dissection begins in the ascending aorta and consists in creation of a re-entry passage from the false to the true aortic lumen. This is done by cross-clamping the descending thoracic aorta, dividing it completely between clamps, obliterating the false passage below by approximating the outer and inner layers, excising a small segment from the inner wall above to permit re-entry into the true lumen and then completing the procedure by end-to-end anastomosis. Blood flow is then directed from the double aortic lumen above into the single normal lumen below.

The second method is indicated in cases in which the dissection begins near the origin of the left subclavian artery and may be considered a more curative procedure, since it removes the origin of the dissecting process and obliterates the false passage. It consists in excision of the segment involved in the origin of the dissection and replacement with an aortic graft after the distal false passage has been obliterated by suturing together the edges of the inner and outer walls. This type of dissecting aneurysm has been the one most commonly encountered in our experience and for this reason this type of repair has been the one most frequently applied. This may be due to the fact that the type in which the dissecting process arises in the ascending aorta tends to be more rapidly fatal, whereas those in the descending thoracic aorta tend to be more slowly progressive.

Analysis of our experience with these methods of treatment has been highly gratifying. Hypertension appears to be one of the most important factors contributing to the risk of operation, since in our series of cases there were no deaths among the patients operated upon that were normotensive (Table I). Follow-up observations for approximately four years have indicated maintenance of good functional activity in the great majority of patients.

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* Med. Research Council, Special Report Series 193, London, H.M.S.O., 1934.

